NKs enter Zone 1 in response to Dendritic Cell agent (DC) activation by parenchymal kinase-1 (PK1) and production of (monokine-1) MK1. A Portal Agent in Zone 1 initially senses MK1, and sends in NKs (NumNKToSend), simulating a chemotactic response [2, 42, 102]. They move randomly until they sense PK1, then they transition to State 1 and follow the PK1 gradient to seek out any stressed Parenchymal Cell agents (PCs) that are producing it. They also produce cytokine-1 (CK1), a pro-inflammatory signal (representing IFN-γ) [97, 103]. The CK1 production is enhanced by MK27 [17]. Although NK cell recognition of self major histocompatibility complex (MHC) Class I on cells provides an inhibitory signal to prevent killing, in a pro-inflammatory environment [29] or if a cell is virally infected [105], the inhibition is overcome. If the NK finds a PC that is stressed and the (pro-inflammatory) PK1 signal present is greater than the CK2 signal, the PC is killed and the NK returns to State 0. The NKs have a limited lifetime (LIFE_NK_Zone1) and they have a limited number of kills that they may execute (NK_KILL_LIMIT) [101].